

Suppressing traffic-driven epidemic spreading by edge-removal strategies

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The interplay between traffic dynamics and epidemic spreading on complex networks has received increasing attention in recent years. However, the control of traffic-driven epidemic spreading remains to be a challenging problem. In this Brief Report, we propose a method to suppress traffic-driven epidemic outbreak by properly removing some edges in a network. We find that the epidemic threshold can be enhanced by the targeted cutting of links among large-degree nodes or edges with the largest algorithmic betweenness. In contrast, the epidemic threshold will be reduced by the random edge removal. These findings are robust with respect to traffic-flow conditions, network structures and routing strategies. Moreover, we find that the shutdown of targeted edges can effectively release traffic load passing through large-degree nodes, rendering a relatively low probability of infection to these nodes.

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Human society has always suffered from various viruses, such as AIDS, H1N1 influenza and computer virus. As the rapid development of complex network theory [1, 2], much effort has been dedicated to understand dynamical processes of epidemic spreading on complex networks in the past decade [3–11]. Propagation is usually assumed to be driven by reaction processes, in the sense that every infected node transmits diseases to all its neighbors at each time step, producing a diffusion of the epidemics in the population. However, in many realistic situations, even when there is a link connecting two nodes, infection will not propagate unless some kind of traffic happens between the nodes. For example, a computer virus can spread over Internet via email-exchanges. In the absence of such data packet transmission, even if there is a path linking two computers, an infected computer will not be able to infect the other one. Another example is that air transport tremendously accelerates the propagation of infectious diseases among different countries.

The first attempt to incorporate traffic into epidemic spreading is based on metapopulation model [12–19]. This framework describes a set of spatially structured interacting subpopulations as a network, whose links denote the traveling path of individuals across subpopulations. Each subpopulation consists of a large number of individuals. Recently, Meloni *et al.* proposed another traffic-driven epidemic spreading model [20], in which each node of a network represents a router and the epidemic can spread between nodes by the transport of information packets.

One of the most important issues in the study of epidemic spreading is how to control the prevalence of infection. To suppress the traffic-driven epidemic spreading, a variety of strategies have been considered, such as the restriction of traffic flow [21], the selection of routings [22] and heterogeneous curing rate [23], etc. In this Brief Report, we propose a method to control traffic-driven epidemic spreading based

on edge-removal strategies. The principle of edge-removal strategies is to affect the spreading dynamics of epidemics (or virus) by deleting some edges in the underlying network. It has been recognized that edge-removal strategies can greatly influence the dynamics of synchronization [24], evolutionary games [25] and traffic [26]. In Ref. [27], Zhang *et al.* found that, both random and targeted deletion of edges can suppress the outbreak of reaction-based epidemic. In contrast, we will show that random and targeted edge-removal strategies play different roles in the traffic-driven epidemic spreading. Specifically, we have found that the random shutdown of edges decreases the epidemic threshold, while the targeted shutdown of edges increases the epidemic threshold.

Following the work of Meloni *et al.* [20], we incorporate the traffic dynamics into the susceptible-infected-susceptible model [28] of epidemic spreading as follows. In a network of size N , at each time step, λN new packets are generated with randomly chosen sources and destinations, and each node i can deliver at most C_i packets toward their destinations. Packets are forwarded according to a given routing algorithm. The queue length of each agent is assumed to be unlimited. The first-in-first-out principle applies to the queue. Each newly generated packet is placed at the end of the queue of its source node. Once a packet reaches its destination, it is removed from the system. Nodes can be in two discrete states, either susceptible or infected. After a transient time, the total number of delivered packets at each time will reach a steady value. Subsequently, an initial fraction of nodes ρ_0 is set to be infected (we choose $\rho_0 = 0.1$ in our numerical experiments). The infection spreads in the network through packet exchanges. All packets queuing in an infected node are infected, while all packets in a susceptible node are uninfected. A susceptible node has the probability β of being infected every time it receives an infected packet from any infected neighboring nodes. With probability $1 - \beta$, the virus in an infected packet will be cleaned by antivirus software in the susceptible node. The infected nodes are recovered at rate μ (here, we set $\mu = 1$).

In the following, we carry out simulations systematically by

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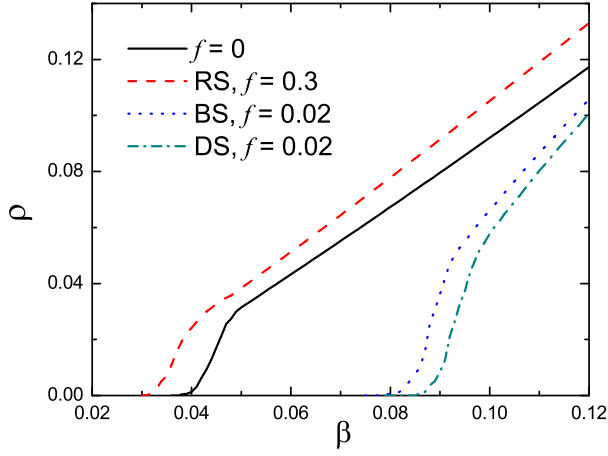


FIG. 1: (Color online) Density of infected nodes ρ as a function of the spreading rate β for the null ($f = 0$), RS ($f = 0.3$), BS ($f = 0.02$) and DS ($f = 0.02$) cases. The packet-generation rate $\lambda = 1$. Each curve is an average of 10^2 different realizations.

employing traffic-driven epidemic spreading on the Barabási-Albert (BA) scale-free networks [29] with the shortest-path routing algorithm [30, 31]. Initially, the size of BA network is set to be $N = 5000$ and the average degree of the network $\langle k \rangle = 10$. Moreover, we assume that the node delivering capacity is infinite, so that traffic congestion will not occur in the network.

Three edge-removal strategies are considered respectively. (I) The random strategy (RS): We randomly remove a fraction f of edges from the network. (II) The betweenness-based strategy (BS): We rank the edges in descending order according to their algorithmic betweenness. The algorithmic betweenness of an edge is the average number of packets passing through that edge at each time step in the steady state. We close a proportion of edges at the top of the ranking list. (III) The degree-based strategy (DS): We define the significance G_{ij} of an edge by the product of the degrees of two nodes i and j at both sides of the edge, i.e., $G_{ij} = k_i \times k_j$. After computing the significance of all edges, we rank the edges in descending order according to their significance. A proportion of edges at the top of the ranking list are removed from the network. For all three strategies, disconnected networks are avoided.

Figure 1 shows the density of infected nodes ρ as a function of the spreading rate β for the null ($f = 0$, i.e., no edges are shutdown during the epidemic spreading process), RS, BS and DS cases. We observe that for each case, there exists an epidemic threshold β_c , beyond which the density of infected nodes is nonzero and increases as β is increased. For $\beta < \beta_c$, the epidemic goes extinct and $\rho = 0$.

Figure 2 shows the ratio of $\beta_c(f)$ to $\beta_c(0)$ as a function of the fraction of deleted edges f for the cases of RS, BS and DS. Here $\beta_c(0)$ is the epidemic threshold for the null case and $\beta_c(f)$ represents the epidemic threshold under the condition that a fraction f of edges in the network are deleted. From Fig. 2, we can see that $\beta_c(f)/\beta_c(0) > 1$ and $\beta_c(f)/\beta_c(0)$ in-

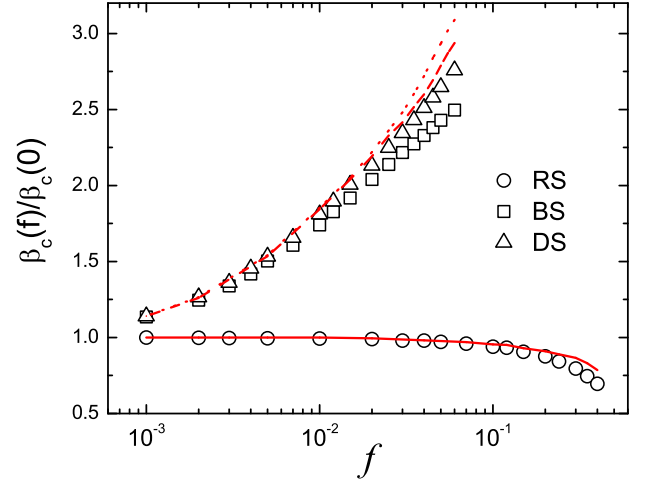


FIG. 2: (Color online) The relative ratio $\beta_c(f)/\beta_c(0)$ as a function of the fraction of deleted edges f for RS, BS and DS. The packet-generation rate $\lambda = 1$ and $\beta_c(0) \simeq 0.043$. Each data point results from an average over 10^2 different realizations. The curves are the theoretical predictions according to Eq. (1) and Eq. (2). The solid, dashed, and dotted curves correspond to the theoretical predictions for RS, BS and DS, respectively.

creases with the increment of f for the cases of BS and DS, indicating that targeted edge-removal strategies can effectively suppress the outbreak of epidemic. As shown in Fig. 2, compared with that of the null case, the epidemic threshold can be enhanced more than 50% when only one percent of targeted edges are cutting-down. It is also noted that the epidemic threshold in the case of DS is a little larger than that in the case of BS, given that the same fraction of edges are deleted. For RS, however, $\beta_c(f)/\beta_c(0)$ is found to decrease as f increases, demonstrating that the random edge-removal strategy is failed to inhibit the spreading of epidemic, but rather enhances its propagation.

According to the analysis of Ref. [20], the epidemic threshold for uncorrelated networks is

$$\beta_c = \frac{\langle b_{\text{alg}} \rangle}{\langle b_{\text{alg}}^2 \rangle} \frac{1}{\lambda N}, \quad (1)$$

where b_{alg} is the algorithmic betweenness of a node [32, 33] and $\langle \cdot \rangle$ denotes the average of all nodes. The algorithmic betweenness of a node is the number of packets passing through that node when the packet-generation rate $\lambda = 1/N$ [32, 33]. For the shortest-path routing protocol, the algorithmic betweenness is equal to the topological betweenness ($b_{\text{alg}} = b_{\text{top}}$) and $\langle b_{\text{alg}} \rangle = \langle D \rangle / (N - 1)$, where $\langle D \rangle$ is the average topological distance of a network. Here, the topological betweenness of a node k is defined as

$$b_{\text{top}}^k = \frac{1}{N(N-1)} \sum_{i \neq j} \frac{\sigma_{ij}(k)}{\sigma_{ij}}, \quad (2)$$

where σ_{ij} is the total number of shortest paths going from i to j , and $\sigma_{ij}(k)$ is the number of shortest paths going from i to j

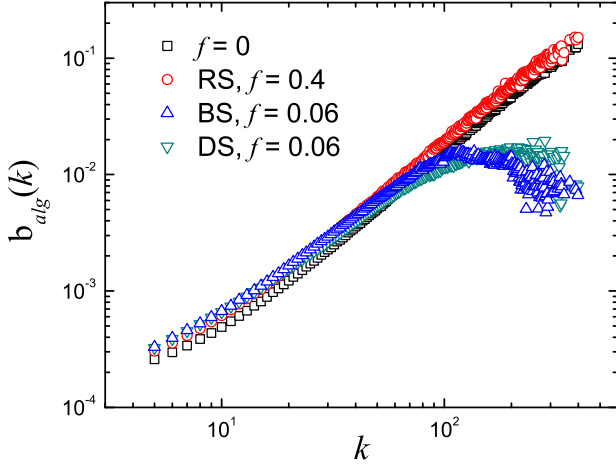


FIG. 3: (Color online) The algorithmic betweenness $b_{\text{alg}}(k)$ as a function of the original degree k for the null ($f = 0$), RS ($f = 0.4$), BS ($f = 0.06$) and DS ($f = 0.06$) cases. Each data point results from an average over 10^2 different realizations.

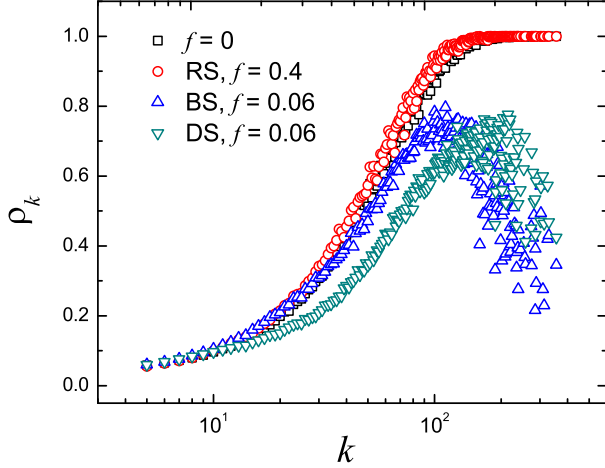


FIG. 4: (Color online) The dependence of the density of infected nodes ρ_k on the original degree k for the null ($f = 0$), RS ($f = 0.4$), BS ($f = 0.06$) and DS ($f = 0.06$) cases. In all cases, the packet-generation rate $\lambda = 1$ and the density of infected nodes $\rho \simeq 0.1$. Each data point results from an average over 10^2 different realizations.

and passing through k . The average topological distance of a network is given by $\langle D \rangle = \sum_{i \neq j} d_{ij} / [N(N-1)]$, where d_{ij} is the shortest distance between i and j . Combining Eq. (1) and Eq. (2), we are able to calculate the theoretical values of $\beta_c(f)/\beta_c(0)$. In Fig. 2, we notice that the theoretical predictions agree well with the numerical results.

To show how different edge-removal strategies affect traffic flow on the nodes with different degrees, we display in Fig. 3 the dependence of algorithmic betweenness $b_{\text{alg}}(k)$ on degree k . Here the degree of a node is calculated before the implementation of deleting edges. From Fig. 3, one can see that for

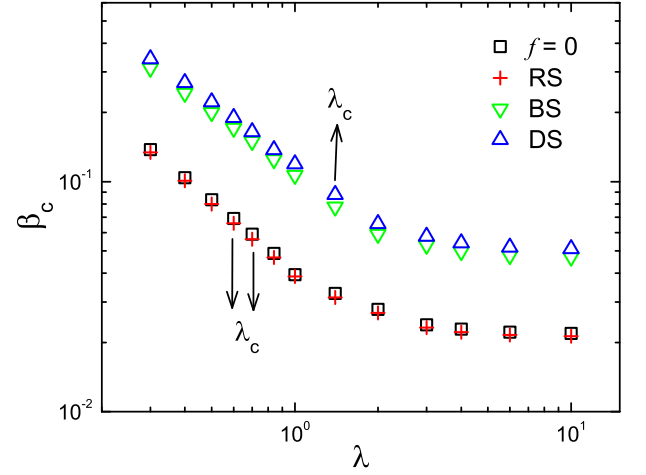


FIG. 5: (Color online) The epidemic threshold β_c as a function of the packet-generation rate λ for the null case ($f = 0$), the RS case ($f = 0.06$), the BS case ($f = 0.06$) and the DS case ($f = 0.06$). For all cases, the delivery capacity of a node i is equal to its original degree, that is $C_i = k_i$. The arrows mark the critical packet-generating rates λ_c . For the null case ($f = 0$), $\lambda_c \approx 0.7$; For the RS case ($f = 0.06$), $\lambda_c \approx 0.6$; For the BS and DS cases ($f = 0.06$), $\lambda_c \approx 1.4$. Each data point results from an average over 10^2 different realizations.

both the null and RS cases, $b_{\text{alg}}(k)$ increases as the increasing of k , and the relationship between $b_{\text{alg}}(k)$ and k follows a power-law form as $b_{\text{alg}}(k) \sim k^\nu$. The exponent ν is almost the same for the null and RS cases. In addition, we can also observe that, for large values of k , $b_{\text{alg}}(k)$ is much smaller in the cases of BS and DS as compared to that in the cases of null and RS. This point is understandable, since the targeted deletion of edges makes many transport paths bypass large-degree nodes and reroute via moderate-degree nodes, hence decreasing the algorithmic betweenness of those hub nodes. Consequently, as shown in Fig. 3, the highest values of $b_{\text{alg}}(k)$ are referred to those medium-degree nodes in the cases of BS and DS.

We define ρ_k as the density of infected nodes of degree k . Figure 4 features the dependence of ρ_k on k for the null, RS, BS and DS cases. Combining Figs. 3 and 4, we can observe that the algorithmic betweenness is positively correlated with the risk of being infected. As shown in Fig. 4, ρ_k increases as k increases for the null and RS cases. Compared with these two cases, the probability of being infected for large-degree nodes is greatly reduced in the cases of BS and DS.

We now turn our attention to a more realistic situation where the node delivering capacity is finite. The main difference with the infinite-capacity case is the possibility of the emergence of traffic congestion in the network, which occurs when the packet-generation rate exceeds a critical value λ_c [33]. Specially, we set the delivery capacity of a node i to be equal to its original degree, that is $C_i = k_i$. The epidemic threshold β_c as a function of the packet-generation rate λ for the null, RS, BS and DS cases are depicted in Fig. 5. We see that β_c decreases and stabilizes at a constant value as λ

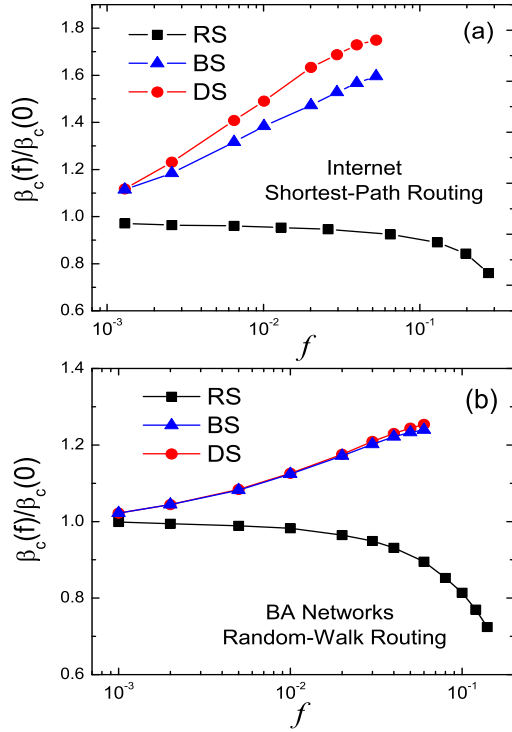


FIG. 6: (Color online) (a) The relative ratio $\beta_c(f)/\beta_c(0)$ as a function of the fraction of deleted edges f for the RS, BS and DS cases. The Internet at the autonomous system level and the shortest-path routing are applied. The packet-generation rate $\lambda = 0.25$ and $\beta_c(0) \simeq 0.045$. (b) The relative ratio $\beta_c(f)/\beta_c(0)$ as a function of the fraction of deleted edges f for the RS, BS and DS cases. The BA networks and the random-walk routing are used. The packet-generation rate $\lambda = 0.02$ and $\beta_c(0) \simeq 0.058$. For both (a) and (b), the node delivering capacity is infinite. Each data point results from an average over 10^2 different realizations.

increases. We also observe that, the random-deletion strategy cannot increase the epidemic threshold while the targeted-deletion strategies (BS and DS) can effectively enhance the epidemic threshold, regardless of that the traffic is in the free-flow state ($\lambda < \lambda_c$) or in the congested state ($\lambda > \lambda_c$).

Finally, we examine the performance of our proposed

strategies in controlling epidemic spreading by considering our model on different network structures and with alternative routing protocols. In Fig. 6(a), we present the simulation results for different edge-removal strategies on the Internet at the autonomous system level [34], where the network size $N = 6474$ and the average degree $\langle k \rangle = 3.88$ before cutting edges. The packets are delivered following the shortest-path routing. In Fig. 6(b), we carry out our studies on the BA networks, where packets are forwarded according to a random-walk routing algorithm, i.e., a packet is delivered to a randomly selected neighbor. For the random-walk routing, the algorithmic betweenness b_{alg} of a node is proportional to its degree [35, 36]. As shown in Fig. 6, the conclusion that the suppression of epidemic outbreak by the targeted edge-removal strategies and the promotion of epidemic outbreak by the random edge-removal strategy, is still valid.

In conclusion, we have studied the impact of edge-removal strategies on traffic-driven epidemic spreading. The shutdown of links in terms of their algorithmic betweenness or of links connecting large degree nodes, are found to be quite efficient in suppressing epidemic spreading. Contrary to previous studies on reaction-based epidemic [27], we find that the random shutdown of edges accelerates the outbreak of traffic-driven epidemic. Furthermore, compared to the deletion of edges with the largest algorithmic betweenness, the shutdown of links connecting large-degree nodes is proved to be more effective in enhancing the epidemic threshold. Thus, according to our present studies, the targeted link-closing method can be used to control the spreading of computer virus in the Internet. For example, we can temporarily close links between large-degree nodes at the time of virus outbreak, and recover these links after virus is eliminated from the system, which could be realized readily by special softwares.

Acknowledgments

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